Lead Exposure and Child Behavior

ABSTRACT

Objectives. Unlike cognitive impairments associated with lead exposure, lead-associated child behavior problems have been difficult to specify, particularly in young children.

Methods. The Child Behavior Checklist (CBCL) and the Center for Epidemiologic Studies Depression Scale were used as the outcome and confounding variables, respectively, of major interest. These measures were examined with respect to blood lead levels of 201 African-American children aged 2 through 5 years.

Results. In comparison with the low exposed group, the high exposed group (two consecutive blood lead levels ≥15 μg/dL) had a significantly higher mean CBCL Total Behavior Problem Score (TBPS) and Internalizing and Externalizing scores; when other factors, including maternal depressive symptomatology, were controlled for, regression procedures indicated a .18-point TBPS increase for each unit increase in lead and a 5.1-point higher TBPS in the high exposed group; children in this group were 2.7 times more likely to have a TBPS in the clinical range.

Conclusions. Through its use of a standardized parent-report measure of behavior and its consideration of maternal morale in multiple linear and logistic regression procedures, this study provides further evidence of lead's detrimental effect on child behavior at levels typical of present-day exposure. (*Am J Public Health.* 1992;82:1356–1360)

William G. Sciarillo, ScD, MSN, Greg Alexander, ScD, MPH, and Katherine P. Farrell, MD, MPH

Introduction

Lead poisoning is the most important pediatric environmental health problem in the United States.[1] Disturbances in neurological and systemic functions have been identified at levels that were once thought not to be cause for concern (i.e., 10 to 25 μg/dL).[2] There is strong evidence that low-level exposure impairs cognitive development,[3] and long-term effects of childhood exposure have been reported.[4]

Lead-associated behavioral difficulties in young children are insidious and difficult to specify. Nonspecific symptoms (e.g., overactivity, irritability, lethargy) may not be recognized until the child enters school. In this study we examine the influence of lead exposure on maternal-reported behavior in children aged 2 through 5 years.

Because maternal morale may color salient aspects of the early caregiving environment, affecting lead exposure (related to compromised caretaking),[5–8] behavioral development,[9] and maternal perceptions of child behavior,[10] maternal depressive symptomatology is considered in this study as a potentially important confounding factor. Low-income women who live alone with their children are at greater risk for depression.[11,12]

Methods

A convenience sample of 150 mother-child dyads from the Baltimore Soil Lead Abatement Demonstration Project's clinic were enrolled in this study during the period from February through April 1989; 54 dyads from the Kennedy Krieger Institute's Lead Poisoning Referral Center were recruited at their clinic visits in April, May, and June 1989. Recruitment from both a population-based study and a clinical facility for referred children ensured a broad range of lead exposure. All subjects met the following enrollment criteria: (1) the child was between 2 and 5 years old; (2) the mother or female guardian accompanied the child to the clinic; and (3) both were African-American. There were three refusals to participate (Kennedy Institute), resulting in a sample of 201 dyads.

Lead concentrations were determined from venous samples collected during the clinics. Children with a blood lead level greater than or equal to 15 μg/dL on two consecutive occasions—at the clinic appointment immediately prior to enrollment in this study and at the first appointment during the study period—were considered the high exposed group. Consistent with preestablished criteria,[13] children with a free erythrocyte protoporphyrin level greater than or equal to 35 μg/dL and a blood lead level less than 25 μg/dL were considered iron-deficient (in the absence of lead poisoning).

Child behavior was measured by interviewing the mother during the child's clinic visit. The Child Behavior Checklist

At the time of the study, William G. Sciarillo was with Children's Medical Services, Local and Family Health Administration, Department of Health and Mental Hygiene, Baltimore, Md. Greg Alexander is with the Maternal and Child Health Program, School of Public Health, University of Minnesota, Minneapolis, Minn. Katherine P. Farrell is with Community Health Services, Anne Arundel County Health Department, Annapolis, Md.

Requests for reprints should be sent to William G. Sciarillo, ScD, Executive Director, Association for the Care of Children's Health, 7910 Woodmont Ave, Suite 300, Bethesda, MD 20814.

This paper was submitted to the Journal June 18, 1991, and accepted with revisions April 27, 1992.

(CBCL),[14–16] a standardized scale designed to quantify troublesome parent-reported behaviors, was used. The CBCL is widely accepted as a reliable and valid approach to behavioral assessment.[17]

Maternal depressive symptomatology was measured by interview; the Center for Epidemiologic Studies Depression Scale (CES–D),[18] a 20-item index of the number and frequency of depressive symptoms,[19] was used. A CES-D score of 16 or more is clinically significant (significance has been validated by correlation with clinical ratings of depression and by associations with negative life events) and may also indicate nonspecific psychological distress.[20,21]

Results

There were 123 children in the low exposed (LE) group and 78 in the high exposed (HE) group. Mean blood lead levels prior to this study were 11.3 μg/dL (SD = 4.3) for the LE group and 28.6 μg/dL (SD = 9.3) for the HE group; mean levels during the study were 9.2 μg/dL (SD = 2.9) and 27.8 μg/dL (SD = 10.4), respectively.

Mean CBCL Total Behavior Problem Scores (TBPSs), Internalizing and Externalizing T-scores, based upon the percentile ranking of raw scores, are presented in Table 1. The HE group consistently had more maternal-reported troublesome behaviors. Prevalence of TBPSs, Internalizing, and Externalizing T-scores in the clinical range (90th percentile) are presented in Table 2. The proportion of HE children with clinical T-scores was approximately double that of the LE group.

The prevalence of subscale T-scores in the clinical range (98th percentile) was examined, providing a more specific perspective of maternal-reported behavior problems. Overall, 17.9% of LE children and 30.8% of HE children had one or more subscales in the clinical range (odds ratio = 2.0; 95% CI = 1.0, 4.0).

The prevalence of clinical T-scores for each subscale of the 2- to 3-year-old (both sexes) profile (n = 118), the 4- to 5-year-olds boys' profile (n = 46), and the 4- to 5-year-old girls' profile (n = 37) is presented in Figures 1 and 2. This prevalence was generally higher in the HE group across all three profiles. For 2- to 3-year-olds and for 4- to 5-year-old boys, this tendency was particularly evident toward the Externalizing end of the continuum of the profiles (e.g., for the Aggressive subscale).

TABLE 1—Mean T-Scores of Low Exposed and High Exposed Groups on the Child Behavior Checklist (CBCL)

	Low Exposed (n = 123)		High Exposed (n = 78)	
CBCL Score	Mean	(SD)	Mean	(SD)
Total*	51.0	(10.5)	55.8	(12.5)
Internalizing**	51.0	(10.2)	55.2	(10.2)
Externalizing***	51.0	(10.0)	54.8	(12.0)

*$t = -2.93$; $df = 199$; $P < .01$.
**$t = -2.89$; $df = 199$; $P < .01$.
***$t = -2.46$; $df = 199$; $P = .01$.

TABLE 2—Prevalence of Clinical T-Scores for Low Exposed and High Exposed Groups on the Child Behavior Checklist (CBCL)

	Low Exposed (n = 123)		High Exposed (n = 78)	
CBCL Score	%	(No.)	%	(No.)
Total*	16.3	(20)	32.0	(25)
Internalizing**	10.6	(13)	19.2	(15)
Externalizing***	14.6	(18)	25.6	(20)

*$\chi^2 = 6.85$; $df = 1$; $P < .01$.
**$\chi^2 = 2.99$; $df = 1$; $P = .08$.
***$\chi^2 = 3.77$; $df = 1$; $P = .05$.

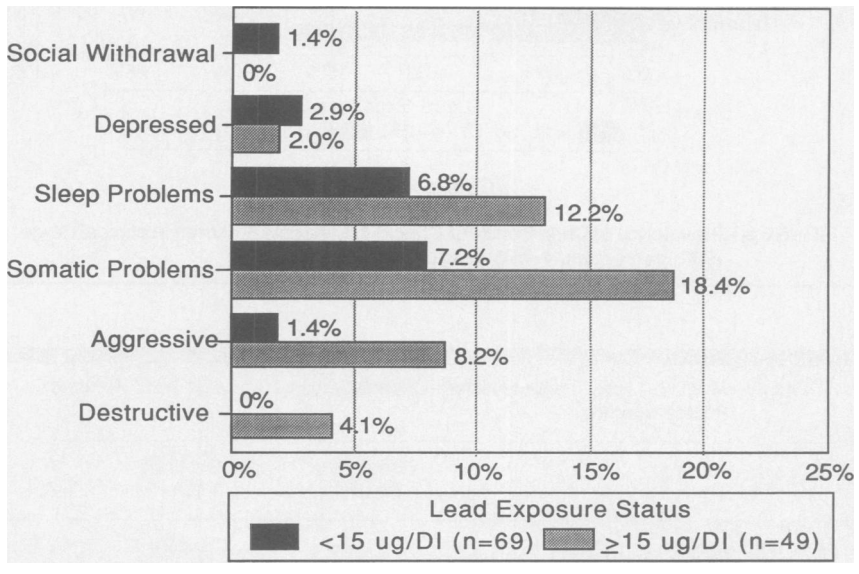

FIGURE 1—Prevalence of Child Behavior Checklist subscale T-scores in clinical range (98th percentile), 2- to 3-year-old boys and girls (n = 118).

Other factors that may have accounted for maternal-reported behavior differences between the two groups were examined. Mean age was 46.4 months (SD = 13.7) for LE children and 44.7 months (SD = 12.9) for HE children ($P = .38$). Maternal education averaged 12.2 years (SD = 1.8) for the LE group and 11.0 years (SD = 1.6) for the HE group ($P < .001$). Mean maternal CES-D scores were 11.3 (SD = 9.9) for the LE group and 13.2 (SD = 9.9) for the HE group ($P = .18$); prevalence of clinical CES-D scores was 25.2% and 34.6%, respectively ($P = .15$). HE children were more likely to come from households in which the mother was not a high school graduate ($P < .001$), was unemployed ($P < .01$), was single ($P < .05$), or was head of the household ($P < .01$), and from

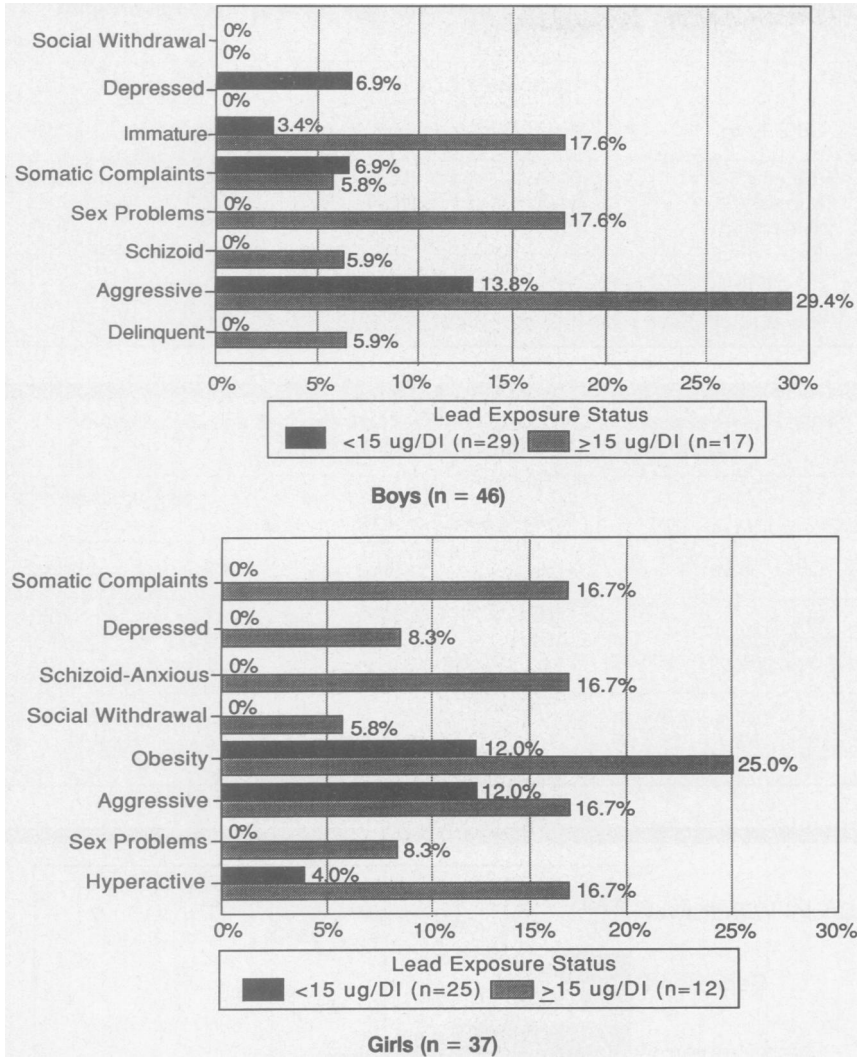

FIGURE 2—Prevalence of Child Behavior Checklist subscale T-scores in clinical range (98th percentile), 4- to 5-year-old boys and girls.

TABLE 3—Multiple Linear Regression on Child Behavior Checklist Total Behavior Problem Score

	Regression Coefficient	Standard Error	*P* Value
Maternal education, y	1.00	0.450	.0279
Maternal occupation: unemployed	2.15	1.614	.1841
Maternal marital status: not married	0.62	1.943	.7505
Preschool children in household: ≥4	−2.78	2.458	.2591
Maternal CES-D score	0.55	0.076	.0001
Child's age, m	0.14	0.054	.0128
Child's sex: male	0.71	1.483	.6334
Iron deficiency[a]	2.95	2.455	.2308
Blood lead, μg/dL	0.18	0.073	.0125

Note. $F = 7.82$, $r^2 = 0.269$.
Intercept = 22.97.

[a]Iron deficiency in the absence of lead poisoning (i.e., free erythrocyte protoporphyrin ≥35 μg/dL *and* blood lead <25 μg/dL).

households in which there was an increased likelihood of four or more preschool-aged children in the household ($P < .05$).

When all other variables in the model were simultaneously controlled for through multiple linear regression, a significant increase ($P = .01$) in the TBPS with increasing blood lead levels was found (Table 3). The magnitude of this effect was quite small (the TBPS increased by .18 points for each unit increase in lead). However, when a dichotomous measure of blood lead (LE vs HE group) was substituted in the model and all other variables in the model were controlled for, a 5.1-point higher TBPS was found in HE children ($P = .001$). Maternal CES-D score was the most significant influence on the child's TBPS ($P = .0001$); child's age and maternal education also had significant independent influence. Similar results were found for the CBCL Internalizing and Externalizing scores.

Multiple logistic regression indicated that HE children were 2.7 times more likely to have a TBPS in the clinical range (Table 4). Maternal depressive symptomatology was most influential (odds ratio = 4.7).

Discussion

Young children with blood lead levels greater than or equal to 15 μg/dL on two consecutive occasions presented more maternal-reported maladaptive behaviors than did children with lower blood lead levels. Initial comparisons between the LE and HE groups indicated significantly higher mean TBPSs, Internalizing scores, and Externalizing scores in HE children. These children also had a prevalence of such scores in the clinical range (90th percentile) that was nearly two times higher than that of children in the LE group.

The HE group generally had a higher prevalence of CBCL subscale scores in the clinical range (98th percentile), especially at the Externalizing end of the continuum of the profiles for 2- to 3-year-old children of both sexes and for 4- to 5-year-old boys. Four- to five-year-old girls in the HE group had a higher prevalence of clinical subscales over the entire Internalizing-Externalizing continuum. The subscales represent factor analytically derived behavioral descriptions, not diagnostic inferences. The prevalence of clinical range subscales for LE children was quite similar to those found in previous reports for young children without known biological risk factors.[15(pp 64,65),22]

TABLE 4—Multiple Logistic Regression on Child Behavior Checklist Total Behavior Problem Score in Clinical Range (90th Percentile)

	Model Level	Adjusted Odds Ratio	95% Confidence Internal
Maternal education, y	<12	0.7	0.3, 1.6
Maternal occupation	Unemployed	1.2	0.5, 2.6
Maternal marital status	Not married	2.0	0.6, 6.4
Preschool children in household	≥4	0.2	0.0, 1.0
Maternal CES-D score	≥16	4.7	2.2, 10.1
Child's age, y	4 or 5	0.9	0.4, 1.8
Child's sex	Male	1.1	0.5, 2.4
Iron deficiency only	Yes	0.8	0.2, 3.4
Blood lead group	High exposed	2.7	1.2, 5.7

Note. Likelihood ratio $\chi^2 = 32.10$; $df = 9$; $P < .001$.

The effect of lead on externalizing behaviors (e.g., increased motor activity) has been demonstrated in animal models.[23–25] The association between exposure and "hyperactive" child behavior has been reported in several case-control studies[26–28] and in Needleman et al.'s population-based study, in which a consistent dose-response relationship between lead and teacher-reported nonadaptive behaviors was found.[29] Others reported children with increased lead levels as demonstrating more behavior problems, particularly with respect to conduct, inattentiveness, passivity, and hyperactivity.[30] A related finding is that hyperactive children have been found to have higher urinary lead levels after penicillamine challenge.[31]

When the influence of selected factors was controlled for in this study through multiple regression analyses, the magnitude of the lead-behavior effect was such that higher lead exposure was associated with a 5.1-point increase in the TBPS. Although this association may not appear to be clinically relevant for an individual child, an overall 5-point upward shift in the normal distribution of TBPSs would have resulted in almost 82% more children scoring in the clinical range. Also, the HE group was 2.7 times more likely to have a TBPS in the clinical range. This finding takes on particular significance in view of the pandemic nature of lead exposure at levels greater than or equal to 15 μg/dL in young children.[2]

The most influential factor on the TBPS in both the multiple linear and logistic regression procedures was the maternal CES-D score. This influence can be explained in at least five ways: (1) mother and child share a similar genotype, (2) mother and child may be reacting to a common factor (e.g., poverty), (3) increased maternal depressive symptoms detrimentally influence child behavior, (4) child behavior problems detrimentally influence maternal morale, and (5) mothers with lower morale rate their children's behavior more negatively. It is reasonable to maintain that some indistinct dynamic combination of all five is operational here. Although the cross-sectional design precludes a more specific delineation, including the maternal depressive symptomatology variable in the regression models provided some degree of empirical control for these possible influences.

With respect to the later possible influence, Conrad and Hammen investigated the relationship of maternal depression to perceptions of internalizing and externalizing disorders in children. They reported that maternal depressive symptomatology and psychiatric clinical status were not associated with misperceptions of child behavior problems.[32] Using teacher ratings as an independent criterion for rating the accuracy of maternal ratings, Richters and Pellegrini found no evidence that depressed mood, depressed clinical state, or history of clinical depression attenuated mother-teacher agreement about child behavior problems.[33]

In addition to the epidemiological significance of an increased prevalence of maternal-reported troublesome behaviors at lead levels typical of present-day exposure, a major clinical implication of this study is that young children with reported behavioral difficulties should undergo appropriate lead screening. Also, the care context in which lead evaluation and treatment occur provides an opportunity to address parental concerns about child behavior. Appropriate interventions, including changing parental misconceptions about normal child behavior, can be initiated. The parent-child dyad is often a captive audience during the lead evaluation and treatment process, and the parent may be more receptive to interventions at this time.

The results of this study, with its particular attention to maternal-reported maladaptive behaviors in young children, lends support to a belief that undue lead exposure in early childhood may have a pervasive influence on the prevalence of juvenile delinquency in this country. □

Acknowledgments

Parts of this study were supported through the Baltimore Soil Lead Abatement Demonstration Project/Maryland Department of the Environment and through the Kennedy Kreiger Institute, Baltimore, Md.

The authors wish to thank Alan Ross, PhD, Vince Smeriglio, PhD, and Mark Farfel, ScD, for their assistance in the design, conduct and analysis of the study, and Julian Chisolm, MD, for his review and discussion of the manuscript (all affiliated with Johns Hopkins University, Baltimore, Md). We also thank Merrill Brophy, RN, MSN, and Marjorie Sheehan, RN, for their assistance in coordinating data collection at, respectively, the Baltimore Soil Lead Abatement Demonstration Project and the Kennedy Kreiger Institute sites.

References

1. Bellinger D, Leviton A, Waternaux C, Needleman H, Rabinowitz M. Low-level lead exposure, social class, and infant development. *Neurotoxicol Teratol.* 1989;10:497–503.
2. *The Nature and Extent of Lead Poisoning in Children in the United States: A Report to Congress.* Washington, DC: Agency for Toxic Substances and Disease Registry; 1988. DHHS publication 99-2966.
3. Needleman HL, Gatsonis CA. Low-level lead exposure and the IQ of children. *JAMA.* 1990;263:673–678.
4. Needleman HL, Schell A, Bellinger D, Leviton A, Allred E. The long-term effects of exposure to low doses of lead in childhood: an 11-year follow-up report. *N Engl J Med.* 1989;322:83–88.
5. Milar CR, Schroeder SR, Mushak P, Dolcourt JL, Grant LD. Contributions of the caregiving environment to increased lead burden of children. *Am J Ment Defic.* 1980;84:339–344.
6. Hunt TJ, Hepner R, Seaton KW. Childhood lead poisoning and inadequate child care. *Am J Dis Child.* 1982;136:538–542.
7. Dietrich KN, Krafft KM, Pearson DT, et al. Contribution of social and developmental factors to lead exposure during the first year of life. *Pediatrics.* 1985;75:1114–1119.
8. Panaccione VF, Wahler RG. Child behavior, maternal depression, and social coercion as factors in the quality of child care. *J Abnorm Child Psychol.* 1986;14:263–278.

9. Zuckerman BS, Beardslee WR. Maternal depression: a concern for pediatricians. *Pediatrics.* 1987;79:110–117.
10. Brody GH, Forehand R. Maternal perceptions of maladjustment as a function of the combined influence of child behavior and maternal depression. *J Consult Clin Psychol.* 1986;54:237–240.
11. Rosenfield S. Sex differences in depression: do women always have higher rates? *J Health Soc Behav.* 1980;21:33–42.
12. Orr ST, James S. Maternal depression in an urban pediatric practice: implications for health care delivery. *Am J Public Health.* 1984;74:363–365.
13. Yip R, Schwartz S, Deinard AS. Screening for iron deficiency with the erythrocyte protoporphyrin test. *Pediatrics.* 1983;72: 214–219.
14. Achenbach TM. The Child Behavior Profile: an empirically based system for assessing children's behavioral problems and competencies. *Int J Ment Health.* 1979;7: 24–42.
15. Achenbach TM, Edelbrock CS. *Manual for the Child Behavior Checklist and the Revised Behavior Profile.* Burlington, Vt: University Associates in Psychiatry; 1983.
16. Achenbach TM, Edelbrock CS, Howell CT. Empirically based assessment of the behavioral/emotional problems of 2- and 3-year-old children. *J Abnorm Child Psychol.* 1987;15:629–650.
17. Achenbach TM, Brown JS. *1989 Bibliography of Published Studies Using the Child Behavior Checklist and Related Materials.* Burlington, Vt: University of Vermont; 1989.
18. Radloff LS. The CES-D Scale: a self-report depression scale for research in the general population. *Appl Psychol Meas.* 1977;1: 385–401.
19. Roberts RE. Reliability of the CES-D Scale in different ethnic contexts. *Psychol Res.* 1980;2:125–134.
20. Weissman MM, Sholomskas D, Pottenger M, Prusoff BA, Locke BZ. Assessing depressive symptoms in five psychiatric populations: a validation study. *Am J Epidemiol.* 1977;106:203–214.
21. Vernon SW, Roberts RE. Measuring nonspecific psychological distress and other dimensions of psychopathology. *Arch Gen Psychiatry.* 1981;38:1239–1247.
22. Larson CP, Pless IB, Miettinen O. Preschool behavior disorders: their prevalence in relation to determinants. *J Pediatr.* 1988; 113:278–285.
23. Sauerhoff M, Michaelson IA. Hyperactivity and brain catecholamines in lead-exposed developing rats. *Science.* 1971;173: 1022–1024.
24. Silbergeld EK, Hruska RE. Neurochemical investigations of low level lead exposure. In: Needleman HL, ed. *Low Level Lead Exposure.* New York, NY: Raven Press; 1980.
25. Ferguson SA, Bowman RE. Effects of postnatal lead exposure on open field behavior in monkeys. *Neurotoxicol Teratol.* 1989;12:91–97.
26. David O, Clark J, Voeller K. Lead and hyperactivity. *Lancet.* 1972;2:900–903.
27. David OJ, Hoffman SP, Sverd J, Clark J, Voeller K. Lead and hyperactivity. Behavioral response to chelation: a pilot study. *Am J Psychiatry.* 1976;133:1155–1158.
28. David O, Clark J, Hoffman S. Childhood lead poisoning: a re-evaluation. *Arch Environ Health.* 1979;34:106–111.
29. Needleman HL, Gunnoe C, Leviton A, et al. Deficits in psychologic and classroom performance of children with elevated dentine lead levels. *N Engl J Med.* 1979;300: 689–695.
30. Lansdown R, Yule W, Urbanowicz M, Miller I. Blood lead, intelligence, attainment and behavior in school children: overview of a pilot study. In: Rutter M, Jones RR, eds. *Lead versus Health.* New York, NY: John Wiley and Sons; 1983.
31. Gittelman R, Eskenazi B. Lead and hyperactivity revisited: an investigation of nondisadvantaged children. *Arch Gen Psychiatry.* 1983;40:827–832.
32. Conrad M, Hammen C. Role of maternal depression in perceptions of child maladjustment. *J Consult Clin Psych.* 1989;57: 663–667.
33. Richters J, Pellegrini D. Depressed mothers' judgements about their children: an examination of the depression-distortion hypothesis. *Child Dev.* 1989;60:1068–1075.